THE RECOVERY KERNEL

DISCONTINUANCE PROTOCOL: OPIOIDS

The withdrawal itself isn't what kills people. What happens afterwards does.

This guide is built around a crucial distinction: getting it wrong can cost lives. Opioid withdrawal, in most cases, will not kill you directly. It's one of the most physically miserable experiences, but it's rarely fatal on its own. What *is* dangerous is what happens afterward: returning to a previous dose after your body has lost its tolerance, making that dose potentially lethal. This single fact changes almost everything else in this guide, and that's why medication-assisted treatment (MAT) is central to rather than at the margins.

Section 0 — Immediate Safety: Read This First

[ ] MEDICAL EMERGENCY

THE HIGHEST-RISK MOMENT IN OPIOID RECOVERY IS NOT WITHDRAWAL. IT IS RELAPSE AFTER A PERIOD OF ABSTINENCE. A person who stops using and later returns to their prior dose is dosing with the tolerance of someone who has never used opioids at all, while expecting the effect of someone who used regularly. This is the single most common mechanism of opioid overdose death, and it applies with even greater force now that the illicit supply is widely contaminated with fentanyl — see Section 6.

Bel immédiatement au 000 et utilisez de la Naloxone si disponible, pour :

- Lent breathing, shallow breathing, or no breathing.
- Blau oder graue Lippen oder Fingerkuppen.
- Incapacité de réagir ou de se réveiller.
- Gurglende eller kvælningslyde under vejrtrækning.

⚠ CAUTION

Naloxone (Narcan) reverses opioid overdose and is available without a prescription at most pharmacies in the US, often free through harm reduction organizations. Having it accessible — for yourself or anyone in your household with any opioid use history, past or present — is not a sign of expecting failure. It is exactly the same category of precaution as a fire extinguisher: something you hope to never need and keep anyway.

[ ] SANCTUARY

The physical symptoms of opioid withdrawal — described in Section 2 — are severe and genuinely miserable, but for most people without other serious health conditions, they are not independently life-threatening. Medical supervision during withdrawal exists primarily to reduce suffering enough that a person doesn't relapse into the danger described above, not because the withdrawal itself typically requires emergency

intervention. Knowing this distinction doesn't make the process easier, but it changes what to actually be afraid of, and when.

Section 1 — Why This Guide Centers on MAT

Medication-assisted treatment – buprenorphine, methadone, or naltrexone – is, by an extensive evidence base, the single most effective intervention available for opioid use disorder. This guide states that directly and without the hedging some recovery material still applies to it: MAT is not a lesser form of recovery, a crutch, or 'still being addicted to something.' It is a life-saving medical intervention with mortality reduction data behind it that few other addiction treatments can match. Recovery work that runs alongside MAT – the biological, environmental, and identity work in Sections 8 and 9 – is additive to it, not an Alternative to it.

THE CENTURION

If you've been told, by a program or a person, that MAT means you 'aren't really sober,' that framing is not supported by the evidence and it costs lives. The measure that matters is survival and functioning, not which specific molecules are or aren't present in a person's system.

Section 2 — The Withdrawal Timeline

Timeline.	What's happening?	What to Expect	What to Do
Hours 6-24 (onset, varies by opioid half-life).	Occupancy of opioid receptors decreasing; the system is no longer receiving the expected stimulus.	Anxiety, muscle aches, sweating, yawning, runny nose, early restlessness.	MAT initiation, if pursued, often begins in this window — buprenorphine specifically requires the person to be in mild-to-moderate withdrawal already before the first dose to avoid precipitating worse withdrawal.
Days 1-3 (peak physical symptoms)	Full withdrawal syndrome active; this is the most difficult physical stage.	Misselijkheid, braken, diarree, buikpijn, verwijde pupillen, rillingen afgewisseld met zweten, ernstige spier- en botpijn, slapeloosheid.	Medical supervision or MAT significantly reduces this severity. This is also the period with the highest risk of someone deciding that the

Timeline.	What's happening?	What to Expect	What to Do
			suffering is unbearable and relapsing to drug use at a now-dangerous dose—see Section 0.
Days 3-10 (post-acute physical resolution, psychological symptoms peaking)	Fysisk abstinens avtar; endorfin- och dopaminsystemen är på sin lägsta punkt.	Depressione profonda, anedonia, forte sensibilità emotiva, insonnia persistente, voglie continue, alcuni sintomi fisici ancora presenti.	La pila completa de suplementos comienza (Sección 9). Haz ejercicio tan pronto como sea físicamente posible, incluso una caminata corta. La gestión del sueño es uno de los predictores más fuertes para evitar la recaída temprana; priorízala directamente.
Weeks 1-4	Sistemi di endorfine e dopamina in lenta fase di recupero; i recettori degli oppioidi iniziano a riattivarsi.	Gradual improvement with real variability day to day; PAWS episodes likely; emotional volatility; episodic but powerful cravings.	Full protocol running. Community connection matters more than it may seem – isolation is a major predictor of relapse in this type of recovery.
Months 1-6	Significativa upregulación de receptores; los sistemas de dopamina y endorfinas se están reconstruyendo activamente; la hiperalgesia inducida por opioides (aumento de la sensibilidad al dolor debido a los propios opioides) está disminuyendo.	Gradual and significant improvements; experiencing authentic moments of enjoyment again; pain sensitivity gradually returning to normal.	Here's where the work transitions from mere survival to becoming sustainable. Identity work (Section 9) is suitable at this stage, and not before.

Timeline.	What's happening?	What to Expect	What to Do
Months 6-18+.	La densité des récepteurs mu-opiacés a considérablement augmenté ; le système endorphine produit à nouveau du confort naturel et favorise les liens sociaux.	Natural enjoyment and connection are accessible without the need for chemical assistance. PAWS (Post-Acute Withdrawal Syndrome) episodes are uncommon. Cravings tend to be situational rather than persistent.	For most people, full endorphin system recovery can be achieved within 12-18 months of sustained recovery, regardless of whether that recovery involves Medication-Assisted Treatment (MAT) or not.

Section 3 — How Long Until the Brain Is Actually Back to Normal.

The time it takes for the brain to fully recover after a traumatic event or substance abuse varies greatly depending on individual factors such as age, overall health, and the severity of the initial injury or addiction. While some individuals may experience significant improvements within weeks or months, others may require years of dedicated recovery and rehabilitation.

It's important to remember that "normal" is subjective and can mean different things to different people. For some, it might mean returning to their pre-trauma cognitive abilities, while for others, it might involve adapting to a new way of life with ongoing support and coping mechanisms.

Here are some general timelines and considerations:

*** **Early Stages (Weeks to Months):** During this period, the brain is actively repairing itself through neuroplasticity, the ability to reorganize neural pathways. This involves forming new connections and strengthening existing ones. Cognitive functions like memory, attention, and executive function may gradually improve as the brain heals. However, it's common to experience lingering symptoms such as fatigue, mood swings, and difficulty concentrating.**

*** **Intermediate Stages (Months to Years):** As the initial healing process subsides, individuals may begin to engage in more structured rehabilitation programs that focus on specific cognitive deficits or emotional challenges. This might involve therapy, medication management, and lifestyle modifications. Progress during this stage can be slower and require consistent effort and commitment.**

*** **Long-Term Recovery (Years):** For some individuals, the effects of trauma or substance**

abuse may persist for years, requiring ongoing support and adaptation. While full recovery to a pre-trauma state might not always be possible, individuals can still achieve significant improvements in their quality of life through continued therapy, self-care practices, and community involvement.

It's crucial to consult with healthcare professionals such as doctors, therapists

Full mu-opioid receptor density recovery, in most people, takes 12-18 months of sustained recovery — a timeline consistent with the other longer-recovery substances in this series. Post-acute withdrawal syndrome (PAWS) specifically can run 18-24 months, and for fentanyl-involved recovery specifically, that window may run longer still, given fentanyl's exceptional receptor-binding potency compared to other opioids.

BEING HEARD

If you're eight months into recovery and still experiencing PAWS waves—sudden anxiety, cravings with no obvious trigger, or periods of feeling emotionally flat—that doesn't mean your efforts aren't working. It simply means you're exactly where you should be in the recovery process. The finish line is often over a year away, and understanding this beforehand is meant to change how you experience a difficult eighth month, not to eliminate those challenges altogether.

Section 4 — What You'll Most Likely Experience

- În primele săptămâni, poate apărea o anhedonie profundă: mâncarea, muzica și persoanele pe care le iubești pot părea lipsite de sens, deoarece sistemele opioide din organism ocupă locurile receptorilor de endorfină și dopamină, ceea ce duce la un nivel biologic scăzut.
- Amplified pain sensitivity (opioid-induced hyperalgesia) — a real, documented phenomenon where the nervous system becomes temporarily more sensitive to pain than it was even before opioid use began, as it recalibrates. This resolves over the timeline in Section 2 and Section 3, but it's disorienting while it's happening.
- Unstable sleep patterns can be a major obstacle in recovery and are often a strong indicator of potential relapse. It's crucial to address sleep issues with the same level of importance as other aspects of your recovery journey.
- Craving sensations that appear without an apparent trigger, especially during periods of Protracted Abstinence Withdrawal Syndrome (PAWS) – this is due to neurological processes and is not indicative of a lack of commitment.
- Un portatori specifici dell'alelo G di OPRM1 possono manifestare un marcato senso di disconnessione sociale o solitudine (Sezione 5): lo stesso recettore

che media la ricompensa oppioide media anche il legame sociale basato sugli endorfine, quindi la sua carenza può sembrare isolamento in sé, non solo desiderio di droga.

Section 5 — Genetic Cross-Reference

Gene	What It Governs	Opioid Recovery Relevance
OPRM1 (A118G)	Mu-opioid receptor structure — the primary receptor that all opioids act on.	THE single most important gene in this entire guide. G-allele carriers have reduced receptor binding affinity — meaning they get less pain relief and less reward from standard doses, which is part of what drove escalation. The same reduced binding also means less endorphin-mediated reward from natural sources (exercise, connection, laughter), which is why natural reward architecture can take longer to feel satisfying for this profile. Critically: G-allele carriers show measurably stronger response to naloxone — this is directly actionable treatment-selection information worth bringing to a prescriber.
CYP2D6	Metabolises codeine, tramadol, and many other opioids into either their active or inactive forms.	Poor metabolizers get little to no effect from codeine or tramadol, which can drive dose escalation chasing an effect that specific drug will never produce for them. Ultra-rapid metabolizers convert codeine to morphine dangerously fast — a documented mechanism of fatality even at standard prescribed doses. Also affects buprenorphine metabolism directly relevant to MAT dosing.
CYP3A4	CYP3A4/5.	The dominant pharmacokinetic gene for the opioids most people in this recovery are actually navigating, including both primary MAT medications. Inhibited CYP3A4 — from genetics or drug interactions (certain antibiotics, antifungals, even grapefruit) — can cause dangerous accumulation of these medications. CYP3A4 inducers can precipitate MAT withdrawal by clearing the medication

Gene	What It Governs	Opioid Recovery Relevance
		too fast. Inform every prescriber of every medication.
ABCB1 (P-glycoprotein)	Trasporto di farmaci attraverso la barriera ematoencefalica.	Variants affect how much opioid crosses into the brain from a given dose — some variants allow greater CNS penetration, producing stronger effects from identical doses and plausibly accelerating dependence development. Also affects buprenorphine and methadone CNS concentration, relevant to MAT optimization.
DRD2 (Taq1A, A1 allele)	Densidade de receptores de dopamina D2.	Circa 30% reduced receptor density in A1 carriers — Reward Deficiency Syndrome. When combined with the OPRM1 G-allele, it produces a compounded reward-system deficit affecting both the opioid and dopamine reward pathways simultaneously — one of the higher-complexity profiles in this series.
COMT	Clearance of dopamine and processing of pain signals.	Relevant specifically for anyone whose opioid use began with legitimate pain treatment — COMT variants affect baseline pain sensitivity independent of opioid exposure, meaning undertreated pain may still need its own dedicated management track (Section 6) even once the opioid dependence itself is addressed.
MTHFR	Metilazione – sintesi dei neurotrasmettitori.	Deficits in baseline dopamine and serotonin synthesis that opioids may have been partially compensating for. 5-MTHF support is foundational, not optional, in this recovery.
GABRA2	GABA-A receptor alfa-2 subunit — tono inhibitorio basal.	Not the primary opioid gene, but a genuine compounding factor, for two reasons: it's the same GABA architecture underlying the common opioid-plus-benzodiazepine or opioid-plus-alcohol co-use pattern (each substance targeting a different receptor system for a related anxiety/inhibition problem), and it drives the specific combined profile

Gene	What It Governs	Opioid Recovery Relevance
		detailed below.

Come status changes what opioids actually feel like.

COMT is an enzyme that breaks down dopamine, a neurotransmitter that plays a role in mood, motivation, and movement. People with certain variations of the COMT gene may experience different effects from opioids.

For example, people with one copy of the "A" allele of the COMT gene tend to be more sensitive to the euphoric effects of opioids, while those with two copies of the "G" allele are less sensitive. This is because the "A" allele leads to higher levels of dopamine in the brain, which can amplify the pleasurable effects of opioids.

However, people with certain COMT variations may also be more likely to experience side effects from opioids, such as nausea and vomiting. This is because higher levels of dopamine can also increase sensitivity to these side effects.

It's important to note that everyone responds differently to opioids, regardless of their COMT status. However, knowing your COMT status can help you understand how you might react to different types of pain medication.

If you are taking opioids, it's important to talk to your doctor about your COMT status and any potential risks or benefits.

COMT regulates how rapidly dopamine and stress-related substances are eliminated from the body, and this elimination rate influences the effects of opioids on an individual—not only affecting the amount used, but also explaining why that effect initially seemed necessary.

COMT Profile	Baseline Before Opioids.	Which specific opioids are being referred to?
Val/Val ('Warrior') — clearance rapide, faible niveau basal de dopamine.	Vaak onderstimuleerd in rust; aangetrokken tot intensiteit en vernieuwing, zelfs zonder het gebruik van middelen.	Opioids provide a different kind of relief than for other profiles — less "finally, quiet" and more "finally, comfortable enough to stop chasing intensity." This profile is somewhat less common in pure opioid dependence than in stimulant dependence, but when present, recovery needs genuine high-intensity outlets, not just calm-focused work.
Met/Met ('Worrier' /	Often displays anxiety or	This is the more common opioid-

COMT Profile	Baseline Before Opioids.	Which specific opioids are being referred to?
'Sage') — clearance lente, tono di base più alto di sostanze chimiche legate allo stress.	hyperactivity even when at rest; the nervous system tends to be overly sensitive, even without substance use.	recovery profile. Opioids' HPA-axis suppression (Section 5's genetic table) directly addresses a chronically overactive stress system — the relief isn't just physical comfort, it's the first time an already-hot nervous system gets to fully stand down. This is also the profile where the HPA-axis rebound described elsewhere in this guide hits hardest, since there's more baseline stress-chemical activity to rebound into overdrive.
Heterozygot (Val/Met).	An intermediate baseline between the two profiles above.	Often a blend – some genuine physical relief-seeking, some stress-system relief-seeking – without either being as extreme as the homozygous profiles.

GABRA2 and the 'Anxious Depressant Pattern'.

This is worth naming directly rather than leaving GABRA2 as a minor footnote in the table above. Your Compendium's own Gene-Substance Matrix identifies a specific combined profile — GABRA2 high-risk plus COMT Met/Met — as the 'Anxious Depressant Pattern,' relevant across alcohol, benzodiazepines, and opioids: compromised self-soothing capacity (COMT) stacked on compromised inhibitory tone (GABRA2), producing the most rapid documented progression to dependence of any combined profile in that matrix.

⚠ CAUTION

If this combination applies to you, two things are worth knowing plainly. First, it's part of why polysubstance use — opioids plus a benzodiazepine or alcohol — is such a common pattern for this specific profile: the opioid is answering the stress-system and pain-relief side, while the GABA-targeting substance is separately answering the underpowered-brakes side, and neither one fully covers what the other is doing. Second, it means recovery for this profile genuinely needs both systems addressed together — the opioid-focused work in this guide, and the GABA-focused work in the Recovery Kernel's Benzodiazepine or Alcohol guides if either substance era också en del av mönstret – istället för att behandla opioidberoendet som hela bilden.

Fenotipificación sin resultados de pruebas.

- Gli oppioidi ti hanno dato sollievo dal dolore quando nient'altro ha funzionato, anche a dosi che altri hanno trovato sufficienti? → Possibile allele G di OPRM1 o variante di ABCB1. È opportuno dare priorità ai test CYP2D6.
- Gli oppioidi ti hanno fatto sentire normale, connesso e a tuo agio in un modo che nient'altro ha raggiunto? → Forte coinvolgimento dell'OPRM1 e una

probabile carenza del sistema degli oppiacei endogeni. Il lavoro di legame sociale (Sezione 9) è centrale, non secondario, per il tuo recupero.

- Did your use begin with legitimate pain treatment? → COMT pain-sensitivity variants may be relevant. Undertreated pain requires its own parallel treatment track — see Section 6.
- È un'esperienza persistente di vuoto o anedonia, indipendente dall'uso di oppioidi? → Probabilmente carenza di recettori DRD2 e coinvolgimento di MTHFR. Il supporto dopaminergico è una priorità assoluta.

Section 6 — Special Populations

Situation.	What makes it different?
Chronic pain as the entry point.	For a significant portion of people in opioid recovery, genuine chronic pain was the original and remains a real, ongoing condition. Recognizing the genetic vulnerability that drove dependence does not mean the pain itself was never real, and it does not disappear when the opioid does. This population needs a pain management track running parallel to, not replaced by, the recovery track — physical therapy, non-opioid pain management, and in some cases buprenorphine specifically prescribed for pain (a legitimate, distinct use case from its role in MAT for dependence) indefinitely alongside recovery work. Unmanaged pain is one of the most predictable pathways back to opioid use of anything in this guide.
Fentanyl-kontaminert forsyning.	The current illicit opioid and, increasingly, illicit stimulant and counterfeit pill supply is widely contaminated with fentanyl, which is dramatically more potent than heroin or prescription opioids by weight. Someone with tolerance built on heroin or pills has no reliable protection against a fentanyl-contaminated batch — the potency differential can be fatal at a dose that would have been unremarkable with the substance they believed they were using. Fentanyl test strips (available through most harm reduction organizations) and naloxone access are not optional precautions for anyone with any current opioid use — they are baseline safety equipment, the same category as Section 0's fire-extinguisher framing.
IV/injection route specifically.	Carries the additional risks documented in the Recovery Kernel's Ears, Eyes, and Teeth guide — injection-site infection, endocarditis, hepatitis C and B, and HIV. Baseline testing for bloodborne infections is appropriate recovery-phase medical care, not an accusation — and modern hepatitis C treatment is curative in the large majority of cases, regardless of how long the infection has been present.

The Substitution and Taper Landscape.

For anyone tapering a prescription opioid specifically, the substance itself changes what the taper actually looks like. This is a landscape overview, not an individual

dosing schedule — the exact numbers require a physician who knows the specific patient, current dose, and duration. What follows is the framework worth bringing into that conversation.

Substance.	**It can be complex to taper for several reasons, including:** * **Underlying health conditions** * **Other medications being taken** * **Withdrawal symptoms** * **Lack of support** * **Difficulty adhering to the tapering schedule** * **Fear of relapse** * **Unrealistic expectations** * **Insufficient medical guidance**	**The General Approach**
Fentanyl (patch or illicit)	Extreme potency means that even small percentage reductions represent large absolute changes; the transdermal patch form adds absorption variability (heat, skin condition, and patch placement all affect delivery) on top of the pharmacology itself.	Patch tapers typically move to the next-lowest patch strength on an extended interval (commonly every 2-4 weeks, following the same percentage-based principle used throughout this library), sometimes with an oral bridge for the gap between patch strengths. Illicit fentanyl tapering is rarely done as a direct patch-to-patch reduction — it usually requires converting to a measurable, prescribable form first (see MAT, Section 8).
Oksikodon de lunga akcije (OxyContin) i drugi opioidi za oralnu primenu sa produženim oslobađanjem.	The extended-release mechanism means that reducing the dose affects both the maximum effect and the length of time it lasts—a decrease that might seem insignificant on paper can result in a noticeable lapse in effectiveness.	Gradual reduction in the extended-release dose, commonly 10-25% every 1-4 weeks depending on duration of use and severity, sometimes with a short-acting formulation available for breakthrough symptoms during the transition — a decision for the prescribing physician, not a self-directed addition.
Hydromorphone (Dilaudid)	Short half-life and high potency relative to morphine mean withdrawal onset is faster and more concentrated than with	Often converted (rotated) to a longer-acting opioid initially to make the tapering process more manageable, rather than tapering

Substance.	It can be complex to taper for several reasons, including: * Underlying health conditions * Other medications being taken * Withdrawal symptoms * Lack of support * Difficulty adhering to the tapering schedule * Fear of relapse * Unrealistic expectations * Insufficient medical guidance	The General Approach
	longer-acting opioids, similar in shape to the short-half-life problem documented for venlafaxine in the SSRI/SNRI guide.	the short-acting form directly—this conversion is a genuine clinical technique, not a workaround, and should be managed by a doctor due to the complex calculations involved.
Tapentadol (Nucynta)	A genuinely different mechanism from the others in this table — dual action as both a mu-opioid agonist and a norepinephrine reuptake inhibitor. This second mechanism means some people tolerate it poorly for reasons that have nothing to do with opioid receptor sensitivity: added norepinephrine activity can produce anxiety, jitteriness, or an elevated heart rate, particularly in anyone already running a high-arousal baseline (the COMT Met/Met profile in Section 5's table is worth considering here specifically).	If tapentadol wasn't well tolerated, that's genuinely useful information for a prescriber — it may point toward the dual mechanism rather than opioid dosing itself as the source of the problem, and toward a different medication choice entirely rather than just a different dose of the same one.

Pharma's Opioid-Sparing Substitute: Gabapentin and Pregabalin.

Un modello di prescrizione comune richiede lo stesso trattamento diretto che questa libreria offre altrove: gabapentina e pregabalin sono spesso offerte specificamente per evitare di prescrivere oppioidi, o per supportare una riduzione della dose di oppioidi coprendo parte del componente di dolore neuropatico. Questo

può essere uno strumento legittimo e utile. Può anche diventare una prescrizione indefinita e non monitorata se nessuno tiene traccia di una data di interruzione.

⚠ CAUTION

The Recovery Kernel's own material on gabapentinoids applies directly here: these are increasingly recognized as carrying real dependence and withdrawal risk of their own, including seizure risk at high doses discontinued abruptly, despite historically being prescribed as though they were risk-free. If gabapentin or pregabalin was offered as an opioid alternative, the same accountability questions from this library's Section on substitute medications apply: what is the stated exit date or review point, is this a time-limited bridge or open-ended maintenance, and what would tapering this specific medication look like if it needs to end. A medication that avoided one dependence Risk is not automatically free of dependence risk itself.

Addressing the underlying cause, not just the taper.

For the significant population in Section 6 whose opioid use began with genuine pain, the taper itself is necessary but incomplete — per Section 1 and Section 6's framing, unmanaged pain is one of the most predictable pathways back to opioid use of anything in this entire guide. Resolving what the pain actually is, and treating it directly, is not a parallel nice-to-have. It's a required part of making the taper hold.

- Una evaluación exhaustiva de la causa del dolor, que puede incluir estudios de imagen, derivación a un especialista o cualquier otro paso diagnóstico que aún no se haya realizado, en lugar de seguir tratando únicamente el síntoma indefinidamente.
- Fisioterapia e gestione farmacologica del dolore senza oppioidi, gestita da uno specialista del dolore insieme al medico che supervisiona la riduzione della dose.
- Ketamine infusion therapy: A real and established option for certain chronic pain conditions that haven't responded to other treatments (complex regional pain syndrome, fibromyalgia, and some types of neuropathic pain). It's administered as a sub-anesthetic infusion in a monitored clinical setting, which is different from how ketamine might be used elsewhere. If standard non-opioid options haven't been effective, it's worth discussing this directly with a pain specialist.

SANCTUARY

If no one has ever determined the cause of the pain—only treated the symptoms—it's important to tell a doctor about this. Reducing the dosage of medication that was managing an unknown issue without also investigating the underlying problem is precisely what this guide aims to prevent.

Section 7 — Use Pattern Classification

Level	Pattern	Risk.	Posture.
1 — Prescritto, de corta duración, sin escalada.	A valid prescription, taken as prescribed and for a short period.	Bassa, ma non nulla: la dipendenza fisica può iniziare entro 1-2 settimane di uso regolare anche senza abuso.	Un plan de réduction progressive, discuté avec le médecin prescripteur, est toujours la meilleure approche, même à ce niveau.
2 — Prescrita, de duración más larga o con una escalada leve.	Uso regular que excede la duración prescrita o aumento gradual de la dosis para mantener el mismo efecto.	Moderate — physical dependence is likely established.	A supervised reduction in dosage is crucial; stopping suddenly can lead to severe withdrawal symptoms, and this is precisely the group of people for whom discussing Medication-Assisted Treatment (MAT), even for a short period, is worthwhile to consider beforehand.
3 — Uso regular sin receta médica o un aumento significativo en la dosis.	Taking more than the prescribed amount, using someone else's medication, or regularly taking illicit drugs without consistent, around-the-clock dosing.	High.	Consultation with medical professionals is highly recommended before attempting to discontinue [substance]. Medication-assisted treatment (MAT) should be considered as an integral component of the process, rather than a measure of last resort.
4 — Uso diario y dependiente, incluyendo para evitar la abstinencia.	Dagelijks of bijna dagelijks gebruik, inclusief gebruik om ontwenningsverschijnselen te voorkomen in plaats van voor het gevoel.	Grave - c'est pour cette population que le protocole de traitement assisté par la méthadone (MAT) et de désintoxication de la section 8	Do not attempt unsupervised cessation. Section 0's relapse-overdose warning applies with full force to this population specifically.

Level	Pattern	Risk.	Posture.
		est conçu.	

Section 8 — Medication-Assisted Treatment and Detox

Medication.	Mechanism.	Clinical Notes
Buprenorphine (Suboxone, Subutex, Sublocade)	Partial opioid agonist — activates the receptor enough to prevent withdrawal and cravings, with a ceiling effect that limits overdose risk compared to full agonists.	Requires the person to be in mild-to-moderate withdrawal already before the first dose (to avoid precipitated withdrawal). Can be prescribed in an office-based setting by trained providers, which meaningfully improves access compared to methadone's clinic-based model.
Methadone.	Full opioid agonist, long-acting — stabilises receptor activity without the peaks and troughs of shorter-acting opioids.	Requires daily dosing at a licensed opioid treatment program in most cases, at least initially. Strong evidence base, particularly for higher-severity or longer-duration dependence. CYP3A4 status (Section 5) affects dosing.
Naltrexone (oral or extended-release Vivitrol).	Antagonista del recettore degli oppioidi: blocca completamente il recettore invece di attivarlo, producendo nessun effetto oppiaceo e bloccando l'effetto di qualsiasi oppiaceo utilizzato in aggiunta.	Requires full detoxification (typically 7-10 days opioid-free) before starting, since it will precipitate severe withdrawal if opioids are still active in the system. OPRM1 G-allele carriers (Section 5) show notably stronger response — genuinely useful, actionable treatment-selection information.

THE CENTURION

La scelta del farmaco MAT è una decisione clinica importante che trae beneficio diretto dalle informazioni genetiche contenute nella Sezione 5: comunicatelo alla persona che sta gestendo questo processo con voi.

Section 9 — The Recovery Protocol

Tier 1 — Biological Foundation (Day 1 onwards)

Support	Role
DLPA (DL-Fenilalanina)	Sistema de endorfinas: soporte previo — comience el día 1 si está comiendo.

Support	Role
NAC (600mg twice daily)	Glutamat och balansering samt minskning av sug.
5-MTHF	Support for foundational methylation and neurotransmitter synthesis.
Magnesium glycinate.	General nervous system support and sleep.
Omega-3 (EPA-dominant)	Algemene neurologische ondersteuning tijdens een periode van ingrijpende systeemherstel.

Tier 2 — Medical

- MAT (Medication-Assisted Treatment) for Section 8, as the primary medical intervention, not an adjunct.
- Gestione del dolore (Sezione 6) da svolgere in parallelo se è presente dolore cronico: questo non è facoltativo per quella popolazione.
- Bloodborne infection testing and treatment if injection use history is present (Section 6).

A Low-Cost Practical Tool: Time-Locked Dispensing.

Worth mentioning because it's inexpensive, genuinely useful, and not often mentioned in recovery material: time-locked medication safes exist specifically for situations like a taper, where the goal is controlled, scheduled access rather than either full self-access or no access at all. A trusted person — a partner, family member, or other overseer — loads the doses according to the taper schedule set with the prescribing physician, and the device physically will not open until the scheduled time, regardless of how the person taking the medication feels in the moment. This directly addresses the same access-and-friction mechanism named elsewhere in Denne bibliotekets afskaffelsesordning: at fjerne muligheden for at handle på en impulsiv trang til en tidlig eller ekstra dosis er i sig selv en reel, fysisk form for støtte, og ikke kun en symbolsk én.

⚠ CAUTION

Standard warnings apply, and they matter: this is a support tool, not a substitute for the medical taper plan itself, and not a substitute for MAT or physician supervision where those are indicated. It requires a genuinely trustworthy, consistently available overseer — the tool doesn't work if the person controlling it isn't reliable, and it can become a source of conflict or resentment if introduced without an honest conversation about why. It does not prevent someone from obtaining medication or substances outside the device entirely, so it addresses one specific risk (impulsive early redosing of the tapering medication) rather than the full picture. Most models have some form of emergency override — know what that is and who controls it before relying on the device. And it should never be the only safety measure in place for anyone in the higher-risk classifications in Section 7 — it's a practical support alongside medical care, not a replacement for it.

Om stoffen van illegale oorsprong: waarom "gewoon doorbijten tot het afbouwen klaar is" op zichzelf al een gevaar is.

This deserves direct, serious treatment rather than a passing mention, because the risk is categorically different from anything else in this guide. A medication from a licensed pharmacy has a documented chain of custody — manufactured under regulated conditions, verified in dose and composition, traceable at every step from production to the bottle in hand. A substance obtained outside that system has none of this. No paperwork, no certification of origin, no verification of what's actually in it, no way to know what it passed through or was combined with before reaching the person taking it. This is not a It's a physical fact about the object itself that matters, regardless of why someone sought it; there should be no moral judgment about the choice to seek it.

MEDICAL EMERGENCY

This is not abstract risk. Per Section 6, the current illicit opioid supply — and increasingly the illicit and counterfeit pill supply generally — is widely contaminated with fentanyl, which is potent enough by weight that a fatal dose can be present without any visible, tastable, or detectable difference from an uncontaminated version. Someone bridging a gap during a taper, or seeking relief from withdrawal symptoms outside medical channels, by using whatever is available on the street or from an unverified source, is not making the same category of decision as continuing a supervised medication. It is a decision made with zero information about what is actually being taken. Fentanyl test strips and naloxone (Section 0, Section 6) reduce but do not eliminate this risk — the only way to fully avoid it is to not use an unverified source at all, which is exactly why the medical and MAT pathways in Section 8 exist as the recommended route through a taper, difficult as that route can be to access.

Tier 3 — Nervous System and Endorphin Architecture (Months 1-12, the central phase).

- Daily vigorous exercise — the main way to boost endorphins without using medication, and the one piece of advice you'll find in all the guides in this series.
- Autenticidad en la comunidad y conexión: no es algo accidental, sino estructural, dada la forma directa en que el sistema de receptores opioides media la recompensa del vínculo social (Sección 5).
- Exposição ao frio, risadas e música — cada um com efeitos reais, embora menores, que estimulam a produção de endorfinas, e que valem a pena serem combinados.

SANCTUARY

La arquitectura de las endorfinas se reconstruye mediante una práctica diaria constante durante meses, no a través de esfuerzos esporádicos. Esto no es una crítica a nadie que haya tenido dificultades para mantenerlo; es la realidad de lo que requiere esta recuperación, y coincide con el cronograma honesto del apartado 3.

Emerging Addiction: Oxytocin and the HPA-Axis Reset.

This section exists because your own Main Book dedicates a full chapter to it, and it deserves an honest, correctly-caveated place here rather than being left out. The mechanism it addresses is real and already named elsewhere in this guide: opioids suppress the HPA axis for as long as they're used, and when they're removed, that suppressed system doesn't return gently to baseline — it rebounds, often dramatically overshooting it. Cortisol floods, the amygdala runs hot, and the nervous system reports an existential emergency with no external cause. This is the physiological reason opioid withdrawal can feel like dying even when it isn't medically dangerous (Section 0) — the felt sense is an accurate report from a genuinely overdriven stress system, not an irrational reaction.

⚠ CAUTION

Oxytocin is a direct antagonist of the cortisol-releasing cascade — it inhibits the HPA axis, functioning as a real off-switch for the alarm system opioids were keeping quiet. Your own source material is explicit and worth repeating with the same honesty: this protocol is experimental, not FDA-approved for addiction treatment, and used currently by only a small number of forward-thinking clinics and informed individuals working at the edge of recovery science. It belongs in this guide as a documented, real mechanism worth knowing exists — not as a self-directed intervention. Anyone considering it should do so with a psychiatrist or a specialist in addiction medicine who is actively involved, maintaining the same high standard as with all other additions at the Emerging tier.

Practically, this is the same category of addition as the intranasal oxytocin material already documented elsewhere in the Recovery Kernel's discontinuance series for benzodiazepine withdrawal — the mechanism (HPA-axis dampening) is the same, and the appropriate caution is the same: real, worth a conversation with a specialist, not yet a standard-of-care tool.

Section 10 — Emergency Recognition, Restated

MEDICAL EMERGENCY

CALL 000 AND USE NALOXONE IF AVAILABLE FOR: slow, shallow, or stopped breathing — blue or gray lips or fingertips — unresponsiveness — gurgling or choking sounds. This applies with particular force after any period of reduced use or abstinence, given the lost-tolerance mechanism in Section 0, and with fentanyl contamination (Section 6) as an added, unpredictable risk layer on any illicit-source opioid.

Section 11 — Full Protocol Cross-Reference Map

Reference	What It Offers
Recovery Kernel Opioid Guide, Section 3	The complete MAT and detox framework that this guide's Section 8 summarises.
Recovery Kernel Fentanyl Guide	Contamination-specific details for Section 6: * Testing strips: [Details about using testing strips to

Reference	What It Offers
	detect contaminants.] * Naloxone access: [Information on how to obtain Naloxone in case of an overdose.] * Pharmacokinetics of fentanyl vs. other opioids: [Explanation of the differences in how fentanyl and other opioids are absorbed, distributed, metabolized, and excreted by the body.]
Recovery Kernel Ears, Eyes, and Teeth Guide, Section 5	Injection-related infection risk detailed in Section 6: [Details about injection-related infection risk would be inserted here if available in the original source.]
Compendium Protocol 14 (Pain Management)	The parallel pain-management track, as mentioned in Section 6, is available for individuals whose opioid use started with legitimate pain treatment.
Compendium Protocol 05 (Environmental Redesign)	Prioridad máxima para este programa de recuperación: los antojos desencadenados por estímulos son el principal mecanismo de recaída en la recuperación de opioides, más que en la mayoría de las otras sustancias de esta serie.
Compendium Gene-Substance Matrix, Appendix A	The direct protocol-code mapping behind Section 5's genetics: GABRA2 + COMT Met/Met ('Anxious Depressant Pattern') maps to P06, P09, P10, P21; GABRA2 alone maps to P01, P06, P20; GABRA2 + MTHFR ('Depleted-Impulsive Pattern') maps to P06, P07, P11, P21. Cross-reference this appendix directly if a full genomic panel is available.
Recovery Kernel Main Book, Section 8 (Oxytocin Protocol)	The full source chapter behind Section 9's Emerging Addition — complete mechanism detail, and the same experimental-use caveats repeated in this guide.
Compendium Protocol 20, 21, C-28 (Identity Architecture)	Appropriate starting around Months 1-6 (Section 2), not before — rebuilding identity separate from the using history.
Recovery Kernel: Complex Trauma and the Substance Connection.	Disturbance related to opioid use often starts during times of significant hardship; trauma-specific interventions shouldn't commence until biological factors are stable and medication-assisted treatment (MAT) is in place—timing is crucial in this context, just like it is throughout this program.

Section 12 — Closing

SANCTUARY

La ripresa dall'uso di oppioidi è una delle poche aree della medicina delle dipendenze con una solida base scientifica comprovata per i suoi trattamenti più efficaci: la terapia farmacologica assistita (MAT) salva vite, ripetutamente e in modo misurabile. Utilizzarla

non è una forma inferiore di ripresa. I sistemi di endorfine e recettori che gli oppioidi occupano possono e si ricostruiscono, secondo un ritmo reale, indipendentemente dal fatto che la ripresa includa o meno farmaci continuativi. Ciò che conta maggiormente, come indicato nella Sezione 0, è superare il periodo in cui la tolleranza e le aspettative non sono più allineate: tutto ciò che si trova in questa guida serve a portarti in sicurezza oltre quel punto.

END OF GUIDE — DISCONTINUANCE PROTOCOL: OPIOIDS

Harm Reduction Resource — Not Medical Advice — Not a Substitute for Emergency Care.

Crisis Resources: 988 (Suicide & Crisis Lifeline, call or text) | SAMHSA: 1-800-662-4357